Original Research

Mortality rates among employees potentially exposed to chrysotile asbestos at two automotive parts factories

Murray M. Finkelstein, PhD, MD, CM

A study of the mortality rates among 1657 employees at two Ontario automotive parts factories that manufactured friction materials containing chrysotile asbestos was initiated in response to the workers' concerns about the effects of asbestos on their health. A total of 1194 men and 258 women had had their first potential exposure at least 10 years before the end of the study period; 563 of the men and 138 of the women had had such an exposure at least 20 years before the end of the study period. A significantly increased rate of death from laryngeal cancer and an elevated rate of death from lung cancer were observed in a cohort analysis. One or two deaths might have been due to pleural mesothelioma. There was no increase in the rate of death from gastrointestinal cancer or from nonmalignant respiratory disease. Case-control analysis showed no association between the risk of laryngeal or lung cancer and the total duration of employment (a surrogate for the extent of ambient exposure to asbestos or other workplace toxic substances) or employment in departments where asbestos had been used. An association between risk of death and occupational exposure is uncertain.

En réponse aux inquiétudes exprimées par les travailleurs quant aux risques sanitaires de l'amiante, on a institué la présente enquête portant sur 1657 employés de deux usines ontariennes où l'on fabrique des pièces de freins à base de chrysotile. La première exposition possible à cette substance remonte à au moins 10 ans avant la fin de la période d'enquête dans le cas de 1194 hommes et 258 femmes et à au moins 20 ans pour 563 des hommes et 138 des femmes. L'analyse par cohortes montre une augmentation de la mortalité par cancer du larynx (celle-ci est significative) et de la mortalité par cancer du poumon, le mésothéliome de la plèvre ayant pu causer un ou deux décès, mais nulle augmentation de la mortalité par cancer digestif ou par maladies respiratoires non néoplasiques. Quant à l'analyse comparative cas-témoins, elle ne permet pas de mettre le risque de cancer du larynx ou du poumon en corrélation ni avec la durée d'emploi, celle-ci tenant lieu d'une mesure de l'exposition professionnelle à l'amiante ou à d'autres toxiques, ni avec le simple fait d'avoir travaillé dans une partie de l'usine où l'on utilise l'amiante. Il n'est donc pas certain qu'il existe un rapport entre l'exposition professionnelle à celui-ci et le risque de décès.

Reprint requests to: Dr. Murray M. Finkelstein, Health Studies Service, Ontario Ministry of Labour, 8th Floor, 400 University Ave., Toronto, Ont. M7A 1T7

Asbestos fibres may induce pulmonary fibrosis and cancer of the lung as well as other sites. Poorly controlled exposures have led to epidemics of disease among Canadian workers, including miners and millers in Quebec and factory workers in Ontario.[1] Studies have generally shown that the risk of asbestos-associated disease depends on the nature of the work and the variety of asbestos mineral used. For example, the processing of chrysotile asbestos in a British friction materials factory was associated with no detectable increase in the mortality rate,[2] whereas the manufacture of gas masks containing crocidolite was associated with a very high risk of death.[3] Authorities in Europe and Canada have responded to the apparent differences in the toxic effects of the various asbestos minerals by adopting different regulatory standards for each mineral. As a result the use of amosite and crocidolite has effectively been eliminated, and chrysotile asbestos has become the sole mineral of any importance in commerce.

In this article the results of a study of mortality rates among workers exposed to chrysotile asbestos in two Ontario factories are presented.

This study was initiated in response to concerns of the workers about the effects of exposure to asbestos on their health. It was recognized that a number of deaths potentially related to asbestos exposure had occurred. Although only a small portion of the workforce had been directly involved in the manufacture of asbestos-containing automotive braking systems, all of the employees believed that they had been exposed to asbestos because of dissemination of fibres throughout the plant.

The factories

The first factory opened in 1929 for the manufacture of automotive component parts. In 1937 the manufacture of drum brake assemblies was introduced. Initially the brake shoes and linings were obtained for assembly already drilled, ground and riveted. In 1940 equipment for drilling, grinding and riveting was purchased; it is from this time that employees may have been exposed to asbestos-containing dust. The brake linings contained chrysotile asbestos in a concentration of 25% to 50%. The balance of the linings consisted of a resin binder and various modifiers and fillers.

In 1963 the brake assembly operation was transferred to a new plant in the same city. In 1969 the drilling, grinding and riveting operations were returned to the older plant, the drum brake assembly operation remaining at the new facility. The factories were closed in June 1980.

Exposure to asbestos

There were only two small brake assembly areas that contained friction materials, and the number of employees in these areas was relatively low. The plant reached its highest employment level, 778 workers, in 1976; only 88 of those employees worked directly with friction materials.

Although the workers in the brake departments had the greatest potential for exposure to asbestos, the others might have been exposed to fibres distributed to other areas of the plant. There is little quantitative information about actual exposure. The first air-sampling survey was performed by government hygienists in 1975; from that time until the factories were closed the concentrations of asbestos were less than 2 fibres/ml of air (the prevailing standard then). The concentrations before 1975 are unknown and may have been higher.

Subjects and methods

The study population comprised 1657 workers (1314 men) who had been employed on or after Jan. 1, 1950, for at least 12 months. A total of 1194 men and 258 women had been hired at least 10 years before the end of the study period; 563 of the men and 138 of the women had been hired at least 20 years before the end of the study period. The distribution of age and duration of employment are found in Table I.

Detailed personnel folders were not available

Table I — Distribution of age and duration of employment among employees potentially exposed to asbestos at two Ontario automotive parts factories

Subjects and variables	Duration of employment, yr			
	1 to < 5	5 to < 20	≥ 20	Total
Main group				
Sex				
Male	523	567	224	1314
Female	226	85	32	343
Mean age, yr*				
Men	27.2	29.7	24.3	27.8
Women	29.0	27.2	25.3	28.2
Mean duration of service, yr				
Men	2.6	11.0	31.0	11.1
Women	2.8	9.8	28.6	6.9
Subgroup†				
Sex				
Male	135	204	224	563
Female	57	49	32	138
Mean age, yr*				
Men	28.0	31.0	24.3	27.6
Women	22.3	24.6	25.3	23.8
Mean duration of service, yr				
Men	2.5	13.2	31.0	17.7
Women	2.9	10.5	28.6	11.6

*At start of study period.
†Those who had had first potential exposure 20 years or more before end of study period.

for all the workers, because the records of workers terminated before 1968 were reduced to summary cards that recorded only such information as name, address and employment dates. This information was abstracted for each member of the group, and detailed work histories were sought later for a case–control study of the causes of death associated with exposure to asbestos.

Vital status was ascertained through record linkage with the Canadian Mortality Data Base at Statistics Canada, Ottawa.[4] Employees not linked to the death registry were presumed to be alive at the end of follow-up, Dec. 31, 1985. The Statistics Canada linkage is a probabilistic procedure; thus, some deaths might have been missed if there was disagreement between the information in the death certificate and the information in the employment record or if any of the deaths that occurred outside Canada were not reported to Canadian authorities. It has been estimated that the standardized mortality ratios (SMRs) would be underestimated by less than 10% because of failure to identify all of the deaths in the cohort.[5]

A standard person-years analysis[6] was done to compare the mortality of the study group with that of the general population of Ontario. Workers 85 years of age or older were withdrawn from the analysis. One-sided p values were computed assuming a Poisson distribution of observed causes of death. Computations for matched case–control analysis were performed using conditional maximum likelihood methods.[7]

Results

Cohort analysis

Tables II and III summarize the numbers of deaths and SMRs among the men and women 20 years or more after their first potential exposure to asbestos. Although the rates differed for certain

Table II — Mortality among men 20 years or more after first potential exposure to asbestos by duration of employment

Cause of death (and ICD* code)	Duration of employment, yr: 1 to < 5 OBS†	1 to < 5 SMR‡	5 to < 20 OBS	5 to < 20 SMR	≥ 20 OBS	≥ 20 SMR	Total OBS	Total SMR
All causes	9	136	17	88	78	112	104	109
Cancer	5	289§	3	64	21	124	29	124
Laryngeal (161)	0	0	0	0	3	1190§	3	854§
Lung (162–163)	3	490§	2	129	6	105	11	140
Gastrointestinal (150–159)	1	193	0	0	5	92	6	81
Other	1	175	1	63	7	125	9	116
Circulatory disease	2	65	10	100	41	114	53	108
Heart disease (410–414)	2	85	5	69	30	112	37	102
Cerebrovascular disease (430–438)	0	0	4	290	5	108	9	142
Respiratory disease (460–519)	1	321	1	83	3	69	5	85

*ICD = International Classification of Diseases.[8]
†OBS = observed no. of deaths.
‡SMR = standard mortality ratio.
§$p < 0.05$.

Table III — Mortality among women 20 years or more after first potential exposure to asbestos by duration of employment

Cause of death (and ICD code)	Duration of employment, yr: 1 to < 5 OBS	1 to < 5 SMR	5 to < 20 OBS	5 to < 20 SMR	≥ 20 OBS	≥ 20 SMR	Total OBS	Total SMR
All causes	5	218	7	167	8	175	20	181*
Cancer	3	308	1	63	2	129	6	146
Lung (162–163)	0	0	0	0	1	555	1	193
Other	3	357	1	72	1	74	5	139
Circulatory disease	1	145	2	129	2	102	5	119
Heart disease (410–414)	0	0	1	106	1	83	2	78
Cerebrovascular disease (430–438)	0	0	1	301	0	0	1	110
Respiratory disease (460–519)	0	0	0	0	1	475	1	205

*$p < 0.05$.

causes of death provincial rates rather than local county rates were used to calculate the SMRs because of the greater statistical stability associated with the former. For example, during the follow-up period in Essex County the all-cause mortality rate was 3% higher than the provincial rate, and the rate of death from cancer was 3% higher among men but 3% lower among women. For men the rate of death from lung cancer was 10% higher and from laryngeal cancer 28% higher than the provincial rates; for women the rate of death from lung cancer was 16% higher than the provincial rate.

Overall mortality rates among the employees were increased among the men and the women. Among the men no diseases except for laryngeal cancer were associated with a significantly increased SMR, and there were no trends of increasing SMR with increasing length of employment. All-cause mortality rates were significantly elevated among the women; the six deaths from cancer were due to lung, skin, breast and ovarian tumours, lymphoma and leukemia.

Three deaths of special interest occurred within 20 years after the first potential exposure to asbestos. One employee died of laryngeal cancer 15 years after being hired; this makes for a total of four deaths from this cause (0.51 deaths expected 10 or more years after first exposure). In the two other cases pleural mesothelioma had been suspected, although the cause of death was recorded as lung cancer. One of these men had begun work in 1952 in the hydraulic assembly department. In 1966 he had undergone chest surgery for biopsy of a pleural mass; the pathologist had diagnosed pleural mesothelioma. The slides from this case have been lost, and it is not possible to confirm the diagnosis. The other man had begun work in 1968 in the brake component assembly department. A tumour in his chest had subsequently developed, and a biopsy specimen had been obtained. The pathologists had been unable to make a firm diagnosis, but the pathology consultant to the Workers' Compensation Board had reported that the morphologic features of the specimen and the distribution of the tumour at the time of surgery were fully consistent with mesothelioma. However, the possibility of metastatic carcinoma could not be excluded. The man died in 1981, less than 13 years after starting work at the factory.

Table IV shows the relation between mortality and time since the first potential exposure among the men. The figures for the women were excluded because of their small numbers and the fact that there had been only one death from lung cancer. The all-cause mortality rate among those employed for 10 years or more was significantly higher after than before 30 years from the first exposure; this was due to increased rates of death from cancer and circulatory disease. The types of cancer that occurred more frequently than expected included laryngeal cancer (three cases [0.19 expected]), renal tumour (two [0.29 expected]) and prostatic cancer (three [0.95 expected]).

Case–control analysis

Because the work histories were not available for all of the subjects a case–control analysis was done to study relations between factors of occupa-

Table IV — Mortality among men by duration of employment and length of time from first potential exposure

Diagnosis	Duration of employment, yr	No. of years since first employment or exposure*: 10 to 19 OBS	10 to 19 EXP†	10 to 19 SMR	20 to 29 OBS	20 to 29 EXP	20 to 29 SMR	≥ 30 OBS	≥ 30 EXP	≥ 30 SMR
All causes	< 10	22	18.8	117	10	8.3	121	4	5.9	67
	≥ 10	32	32.2	99	26	32.3	81	64	48.9	131
	Total	54	51.0	106	36	40.6	87	68	54.8	124
Cancer, all types	< 10	2	4.4	46	5	2.0	249	1	1.7	59
	≥ 10	11	6.9	159	5	7.1	70	18	12.7	142
	Total	13	11.3	115	10	9.1	110	19	14.4	132
Lung cancer	< 10	1	1.4	72	3	0.7	441	1	0.6	161
	≥ 10	3	2.1	145	2	2.2	89	5	5.6	90
	Total	4	3.5	116	5	2.9	172	6	6.2	97
Laryngeal cancer	< 10	0	0.06	0	0	0.03	0	0	0.03	0
	≥ 10	1	0.10	1000	0	0.11	0	3	0.19	1600
	Total	1	0.16	625	0	0.14	0	3	0.22	1300
Gastrointestinal cancer‡	< 10	1	0.9	115	1	0.4	233	0	0.4	0
	≥ 10	3	1.7	179	1	1.8	57	2	2.6	77
	Total	4	2.6	154	2	2.2	91	2	3.0	67
Circulatory disease	< 10	12	7.9	152	3	3.9	76	2	2.9	70
	≥ 10	16	15.5	103	13	17.1	76	35	25.3	138
	Total	28	23.4	120	16	21.0	76	37	28.2	131

*For the interval 10 to 19 years 1194 men were at risk, for 20 to 29 years 505 were at risk, and for ≥ 30 years 283 were at risk. These categories are not mutually exclusive.
†EXP = expected no. of cases.
‡Includes cancer of the esophagus, stomach and bowel.

tional exposure (e.g., employment in a department where asbestos had been used) and the risk of death from asbestos-associated disease. An attempt was made to match each person who had died of lung cancer, laryngeal cancer, gastrointestinal cancer or nonmalignant respiratory disease with three control subjects who had survived to at least the age of the case subject. The control subjects were randomly matched to the index cases for sex and year of birth.

Employment records could not be located for five control subjects, and they contained only pension data and no chronology of job location for eight others. For most of the subjects departmental assignments could be traced. Potential exposure to asbestos was assessed for each person by computing the length of time in departments where asbestos had been used and the length of employment up to 5 years before the death of the case subject or up to 3 years before the diagnosis of laryngeal cancer.

Lung cancer and mesothelioma: The death certificates of 18 people recorded lung cancer as the cause. Additional information was available to support this diagnosis in 15 of the cases and included findings at autopsy (5 cases) and at surgery (4), results of biopsy (4), cytologic features of sputum (1) and findings from radiologic investigation (1). As previously mentioned, 2 of the 18 deaths may have actually been due to pleural mesothelioma.

Two (11%) of the 18 case subjects had never worked in a department where asbestos had been used, as compared with 12 (23%) of the control subjects. When the interval between the time of the first exposure and death was a minimum of 20 years the proportion of exposed cases was similar to the proportion of exposed controls (17% and 23% respectively). Case-control analysis revealed no association between the risk of lung cancer and employment in a department where asbestos had been used or duration of employment.

Laryngeal cancer: Four people had died of laryngeal cancer; all were men who had worked more than 10 years at one of the factories. In each case the diagnosis was made on the basis of histologic evidence of squamous cell carcinoma.

None of the four men had worked in a department where asbestos had been used, although some may have been in these areas carrying out other assignments. One of the men had worked as a maintenance millwright, and another had been head of the cleaning crew when dry sweeping of dust was common. Case-control analysis revealed no significant association between the duration of employment and the risk of laryngeal cancer.

Gastrointestinal cancer and nonmalignant respiratory disease: In the cohort analysis there was no increase in the rate of death from gastrointestinal cancer or nonmalignant respiratory disease. No cases of asbestosis were diagnosed. In the case-control analysis there were no significant differences in the distribution of exposure factors between the case subjects and the control subjects.

Discussion

The cohort analysis revealed increased mortality rates among the male and female employees; there was a significant increase in the rate of death from laryngeal cancer and an elevated rate of death from lung cancer. There was no increase in the rate of death from gastrointestinal cancer or from nonmalignant respiratory disease.

The results are difficult to interpret, and it is unclear whether there is any causal connection between the increased mortality rates and the occupational exposures. Case-control analysis showed no association between the risk of death from laryngeal or lung cancer and the duration of employment (a surrogate for the extent of ambient exposure to asbestos or other workplace toxic substances) or employment in departments where asbestos had been used.

Cigarette smoking is a risk factor for laryngeal cancer and lung cancer; therefore, differences in smoking habits between the factory workers and the general population might have accounted for part of the increased risk. Fragmentary data about smoking habits were collected in a 1981 telephone survey in which responses were obtained from 426 (26%) of the study subjects. Although subject to selection bias, the figures suggested that the smoking pattern among the male employees did not differ greatly from the provincial average.

There have been two larger studies of exposure among workers in friction materials factories. Berry and Newhouse[2] found no detectable increase in the rate of death from lung, gastrointestinal or other forms of cancer in a study of 13 000 workers at a British friction materials factory. A number of cases of mesothelioma had been reported, but these were attributed to exposure to crocidolite, which had been intermittently used for special purposes at the factory. There were two deaths from laryngeal cancer (3.6 deaths were expected).

McDonald and associates[9] found a significant increase in the rate of death from respiratory cancer (49 observed, 35.7 expected, SMR 137); the SMR was similar to that for respiratory cancer in our study (168). However, deaths from laryngeal cancer accounted for 4% of all the deaths from respiratory cancer in their study, as compared with 21% in our study. They found no reports of death from mesothelioma.

There is little information in the literature about health risks among employees of automotive parts manufacturers. The results of studies involving machinists exposed to cutting fluids and abrasive agents have not revealed an increased risk of lung cancer but have suggested the possibility of an increased risk of cancer of the gastrointestinal tract,[10,11] an area not at increased risk in our study.

The four deaths from laryngeal cancer repre-

sent a mortality rate about 10 times higher than the provincial average. None of the men had worked in a department where asbestos had been used, although two may have had direct contact with asbestos dust in their respective capacities as millwright and head of the cleaning crew. There are few established causes of laryngeal cancer, and they involve mainly personal factors such as tobacco smoking and alcohol consumption. Information on these risk factors was generally unavailable for the subjects in our study. Two Canadian case-control studies have attempted to identify occupational causes of laryngeal cancer. A study in British Columbia[12] failed to identify any occupational factors, and a study in Ontario[13] revealed a weak association between laryngeal cancer and exposure to asbestos, foundry fumes and metal dusts but not nickel.

Exposure to asbestos is generally accepted as a cause of laryngeal cancer,[14] although this relation has recently been questioned.[15] Among people who have had occupational exposure to asbestos the risk of death from laryngeal cancer is generally less than the risk of death from lung cancer.[14] Given the diversity of jobs held by the workers in our study who died from laryngeal cancer and the small number of cases it is not possible to draw any firm conclusions about an occupational cause.

In summary, there were increased mortality rates among the employees at two Ontario automotive parts factories. Potentially toxic exposures to chrysotile asbestos, cutting fluids and abrasives occurred, but it has not been possible to determine whether occupational factors were responsible for the increased mortality rates.

References

1. *Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario*, Ontario Ministry of the Attorney General, Toronto, 1984: 171-231
2. Berry G, Newhouse ML: Mortality of workers manufacturing friction materials using asbestos. *Br J Ind Med* 1983; 40: 1-7
3. Jones JSP, Smith PG, Pooley FD et al: The consequences of exposure to asbestos dust in a wartime gasmask factory. In *Biological Effects of Mineral Fibres*, vol 2, publ no 30, International Agency for Research on Cancer, Lyon, 1980: 637-653
4. Howe G, Lindsay J: A generalized iterative record linkage computer system for use in medical follow-up studies. *Comput Biomed Res* 1981; 14: 327-340
5. Finkelstein MM: Mortality among employees of an Ontario asbestos-cement factory. *Am Rev Respir Dis* 1984; 129: 754-761
6. Coleman M, Douglas A, Hermon C et al: Cohort study analysis with a Fortran computer program. *Int J Epidemiol* 1986; 15: 134-137
7. Storer B: *PECAN Statistical Software*, Dept of Biostatistics, U of Washington, Seattle, 1983
8. *International Classification of Diseases*, 9th rev, WHO, Geneva, 1978
9. McDonald AD, Fry JS, Wooley AJ et al: Dust exposure and mortality in an American chrysotile asbestos friction products plant. *Br J Ind Med* 1984; 41: 151-157
10. Decoufle P: Further analysis of cancer mortality patterns among workers exposed to cutting oil mists. *JNCI* 1978; 61: 1025-1030
11. Park RM, Wegman DH, Silverstein MA et al: Causes of death among workers in a bearing manufacturing plant. *Am J Ind Med* 1988; 13: 569-580
12. Elwood JM, Pearson JCG, Skippen DH et al: Alcohol, smoking, social and occupational factors in the etiology of cancer of the oral cavity, pharynx, and larynx. *Int J Cancer* 1984; 34: 603-612
13. Burch JD, Howe GR, Miller AB et al: Tobacco, alcohol, asbestos, and nickel in the etiology of cancer of the larynx: a case-control study. *JNCI* 1981; 67: 1219-1224
14. Doll R, Peto J: *Asbestos. Effects on Health of Exposure to Asbestos*, Health and Safety Commission, London, 1985: 7
15. Chan CK, Gee JBL: Asbestos exposure and laryngeal cancer: an analysis of the epidemiologic evidence. *J Occup Med* 1988; 30: 23-27

Research

To him who devotes his life to science, nothing can give more happiness than increasing the number of discoveries, but his cup of joy is full when the results of his studies immediately find practical applications.

— Louis Pasteur (1822-1895)